SHORT REPORT

Pontomedullary sulcus infarct: a variant of lateral medullary syndrome

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With the advent of magnetic resonance imaging (MRI) technology, it is now possible to identify and determine the precise location of medullary infarcts. The lateral part of the medulla is most commonly affected by infarction. Classifications of lateral medullary infarcts are usually based on anatomical data, using rostrocaudal and dorsoventral axes to establish correlations with clinical symptoms.[1–3] Different subtypes of lateral medullary syndrome, depending on location, shape and size of the infarct, have been described in the literature.[4] We report a rare case of a patient presenting with an unusual clinical picture in relation to an infarct specifically located in the lateral part of the pontomedullary sulcus.

CASE REPORT

The patient was a 62-year-old man, with no history of cardiovascular disease and no risk factors such as smoking, diabetes, dyslipaemia or atrial fibrillation. He developed acute sensory changes over the right upper limb extending towards the lateral area of the neck and was hospitalised 24 h after symptom onset.

Physical examination showed numerous shaving cuts on the right side of the neck and decreased pinprick, temperature and pain sense of the right upper limb and of the right side of the neck. No sensory abnormalities were found in the trunk or lower limbs. The patient had slight peripheral facial paresis and tactile hypoaesthesia of the left face. He did not have headache, dysphagia, vertigo or dizziness, nausea or vomiting, Horner's sign, or gait ataxia. The electrocardiogram, chest roentgenogram and laboratory tests were normal. Arterial blood pressure was 170/90 mm Hg.

Brain MRI showed an infarct involving the left part of the pontomedullary sulcus (fig 1). Magnetic resonance angiography was normal. Transoesophageal echocardiography showed a myxoid aspect of the mitral valve. The aortic arch was normal. Systolodiastolic hypertension necessitated treatment.

During hospitalisation, sensory symptoms and mild facial paresis gradually improved but persisted at discharge (National Institutes of Health Stroke Scale 2). Aspirin had been given during the acute phase and was continued after discharge.

DISCUSSION

Data on infarcts in the lateral pontomedullary junction are limited and have appeared, more often, in studies on infarction in the upper medulla or lower pons.[6] To our knowledge, infarcts specifically located in the pontomedullary sulcus have not yet been reported. The clinical presentation of our patient is more comparable with infarcts reported in the lateral upper medullary region than in the lateral lower pons. Moreover, a lateral medullary infarct is the most common type of brain stem stroke and has been widely discussed in the literature, most often in anatomo-radio-clinical correlation studies.[2–4]

In the present case, the use of three-dimensional MRI confirmed that the infarct was solely located in the left lateral part of the pontomedullary sulcus. The centrobicommissural reference plane was used for the horizontal slices to analyse the images with an appropriate anatomical atlas.[7] Although the three space planes are rarely used in everyday practice,[3] they provide important information about the precise topography of lesions and help to refine correlation studies. In our patient, the lesion was found in the arterial lateral territory of the upper medulla, which is supplied by the superior and posterior rami of the lateral medullary fossa arising from the vertebral artery or the anterior inferior cerebellar artery.[5]

In our patient, sensory disturbances were to the forefront. This is consistent with other papers reporting lateral medullary syndrome.[2 4] On the other hand, the sensory hallmarks of lateral medullary syndrome include a loss of spinothalamic sensation of the ipsilateral face and the contralateral hemibody, as usually seen in Wallenberg's syndrome.[2 4 8 9] MRI or clinical correlation studies have described varying sensory patterns of lateral medullary syndrome.[10] In contrast with what was observed in our patient, sensory signs often affect the trunk, so much so that certain authors have investigated sensory loss at the level of the trunk in lower lateral brain stem lesions.[11] Nevertheless, the elective sensory disorder on the contralateral upper limb and that at the base of the neck, shown in our study have not as yet been reported. Some studies have shown that the upper limb, especially the fingers, can have sensory disturbances, but this is usually associated with the ipsilateral hemibody and more often with lemniscal sensory perceptions.[12]

In our patient, the infarct lesion included the medial part of the spinothalamic tract, resulting in loss of pain and temperature senses of the contralateral upper limb and the base of the neck. These findings confirm the hypotheses that have been put forward on the somatotopia of the spinothalamic tract in its medullary course, with a more medial location of cervical afferent fibres.[13 14] The absence of sensitive epicritic signs correlates well with the location of the infarction, as it did not involve the lemniscal tract in its medullary course.

Ipsilateral tactile hypoaesthesia of the face is, in our patient, the consequence of a lesion affecting the pars oralis of the trigeminal spinal nucleus. This part of the trigeminal spinal nucleus is linked to the pontine nucleus of the fifth nerve. It receives the fibres responsible for tactile sensory perception of the ipsilateral side of the face, which then comes to synapse. Owing to its ventromedial location, the ventral trigeminothalamic tract, made up of fibres that

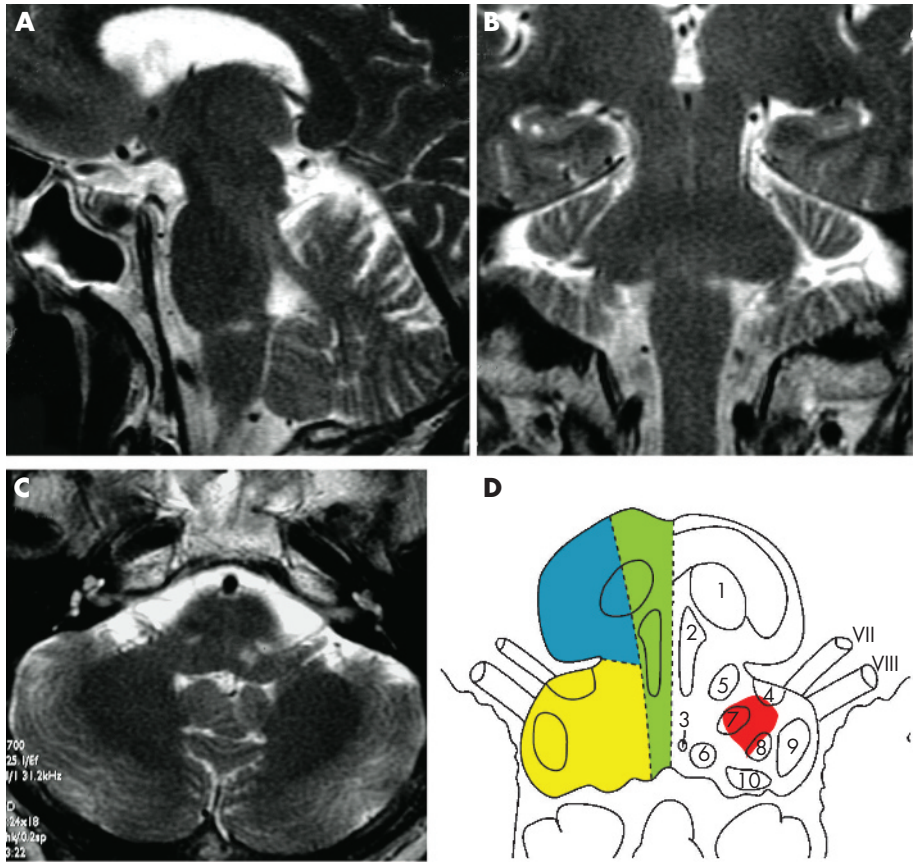

Figure 1 (A) Sagittal and (B) coronal T2-weighted magnetic resonance images (MRIs) showing infarct in the left part of the pontomedullary sulcus. (C) Horizontal T2-weighted MRI and (D) drawing of an axial section of the brain stem at the level of the pontomedullary sulcus (modified after Tatu *et al*[5]). Infarct lesion is drawn in red. (1) Corticospinal tract, (2) medial lemniscus, (3) medial longitudinal fasciculus, (4) spinothalamic tract, (5) inferior olivary nucleus, (6) nucleus prepositus, (7) facial nucleus, (8) spinal trigeminal nucleus, (9) inferior cerebellar peduncle, (10) vestibular nucleus, (VII) facial nerve, (VIII) vestibulocochlear nerve. Medullary arterial territories: anteromedial (green), anterolateral (blue) and lateral (yellow).

ascend towards the thalamus, is spared. The fact that the rest of the spinal nucleus of the fifth nerve is spared explains the absence of thermalgic sensitivity of the other side of the face. These observations confirm the hypotheses already put forward from a previous study on this correlation.[10]

Facial paresis is a less common sign in lateral medullary infarction. Central facial paresis occurs most often in patients with lesions of the lower pons or the upper medulla.[2 4 8 15] Similar to the present study, facial paresis is most often moderate and ipsilateral to the lesion.[2 4 15] As in other cases reported in the literature,[2 4] it can be central[3 15] or peripheral.[3 6] The physiopathology of facial paresis is not well understood. When a central paresis is observed, it is hypothesised that the aberrant lower loop of the cortico-nuclear tract is affected before its projection on the nucleus of the facial nerve.[15–18] When peripheral paresis is reported, as in our patient, this is most often explained by an upper extension of the infarct to the facial nucleus or of the facial fascicle.[6] In our patient, the peripheral facial paresis seemed to be associated more with the infarction affecting a part of the nucleus of the facial nerve than the nerve's intra-axial fascicle. From an anatomical point of view, the nucleus of the facial nerve is visible on the section at the level of the pontomedullary sulcus, whereas the fascicles appear on the superjacent slices. This is another interesting observation that may explain the different presentations of facial paresis.

Our patient did not present with dysphagia or dysphonia, a clear indication that the ambiguous nucleus was spared, which is usually described as being affected by infarcts in the upper medulla.[4] This observation is, perhaps, more specific of a lesion in the pontomedullary sulcus, which represents the upper limit of the pons and is where the ambiguous nucleus is not visible.

In conclusion, the present case illustrates an unusual variant of lateral medullary syndrome with an infarct specifically located in the lateral part of the pontomedullary sulcus. The use of three-dimensional MRI and a detailed anatomical interpretation enabled us to establish accurate anatomoclinical correlations towards a better understanding of the role of anatomical structures in the brain stem.

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